TRAUMATIC HEMATOMYELIA,

WITH A REPORT OF A CASE.

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HEMATOMYELIA is a term applied to a hemorrhage that takes place within the substance of the spinal cord. It is always intra medullary. That we should fully understand this condition, we should briefly review the anatomy of the blood supply of the cord. The blood is supplied by three main arteries and their branches, one anterior and two posterior spinal arteries, all three of which are branches of the vertebral; they are given off from the vertebral near where these join to form the basilar. The two anterior spinal trunks unite in a single artery in front of the medulla, which extends the entire length of the spinal cord in front. It is imbedded in the pia mater and lies in front of the anterior median fissure. The two posterior spinal arteries pass from their origin to the postero-lateral borders of the medulla and proceed separately the whole length of the spinal cord, being located just behind the posterior nerve roots. These arteries are also imbedded in the pia mater. The spinal arteries receive accessory branches along their course from the thyroid, intercostal, lumbar and ilio-lumbar arteries, through the intervertebral foramina, and give off numerous branches to the substance of the cord, through which the cord receives its vascular supply. As before noted, both the anterior and posterior spinal trunks are imbedded in the pia mater, hence are external to the cord. Therefore all hemorrhages that may take place from these trunks will not produce hematomyelia. If the rupture in the arterial wall is so located that the bleeding takes place into the subarachnoid space, hematomyelia does not result. In other words, the hemorrhage must take place in such a manner that the cord substance is invaded by it to produce hematomyelia. In hematomyelia the damage is usually wrought in the gray columns of the cord, these offering the least resistance to the flow of blood, yet in some cases in which the hemorrhage is profuse the white tracts are invaded, but in these cases the blood does not follow the white tracts for any considerable distance, the axones acting as a barrier to its progress.

The ætiological factors in the production of hematomyelia are diseased conditions of the arterial walls; for instance, arteriosclerosis or conditions due to specific disease and traumatism. The former may be present, the latter is always present. The hemorrhage may be in the traumatic area or remote from it, the latter usually prevailing in uncomplicated cases. When the hemorrhage takes place in the traumatic area, the bony wall of the spinal canal is either crushed and splinters of bone pierce the substance of the cord, or it is pierced by knife or bullet wounds. In these cases much damage is wrought to the bony wall, membranes of the cord, and the cord itself, and the hematomyelia thus produced is only a part of the general destruction. While hematomyelia exists in these cases, it is not that uncomplicated form the symptoms of which are at times obscure and the diagnosis difficult, nor that form which we wish to emphasize in this paper.

The form of hematomyelia with which we are dealing takes place in the cervical cord, remote from the traumatic area, and usually follows the violence immediately, but may be postponed for several days or weeks. Its constant site in this class of cases being the cervical region, by reason of the fact of the great mobility of the spinal column in this region. Traumatisms applied to the nates, by falling, or by being struck by ponderous bodies, or applied to the lumbar or dorsal regions producing a sudden exaggerated extension or flexion of the cervical spinal column, may rupture either the anterior or posterior spinal arteries, by over-stretching or by forcibly dragging them against the bony wall of the spinal column. If the arterial walls are diseased the chance for their rupture is greatly enhanced. The traumatic insults in this class of cases may be severe, or so slight as to escape detection by external appearances. In those cases in which the hemorrhage immediately follows the injury, the walls of one or more of the arteries are completely ruptured, but in that class of cases in which the hemorrhage takes place at some rather remote time, as several weeks or two months after the injury, it is probable that at the time of the injury, while the walls of the arteries are not completely ruptured, that their coats are so injured that they later give way, or perhaps only the intima-vasorum is ruptured, an aneurism following, which later ruptures producing the hematomyelia. I believe the above is a reasonable solution of these postponed hemorrhages. These vessels at the point of rupture are subjected to considerable blood pressure, being in quite close proximity to the heart.

As before noted, the damage to the cord is usually confined to the gray columns, and may be much or little according to the amount of bleeding. If the hemorrhage is considerable it will push its way through the gray columns for a distance of three or four segments of the cord above and below the bleeding point. Should the hemorrhage involve the third, fourth and fifth cervical cord segments, serious results will follow, the nerve centres of the phrenic being involved and paralysis of respiration resulting. In the form of hematomyelia of which we are speaking the upper extremities, and the rest of the body below the lesion, will be more or less involved and more or less permanently involved, according to the amount of bleeding that has taken place into the cord.

These hemorrhages may be divided into four categories as follows; local, profuse local, disseminated, and profuse disseminated. The neuroglia tissue in the gray columns of the cord offers greater resisting force to the flow of blood in some cases than in others. Hence in those cases where there is a marked resistance by this tissue, the blood does not make channels between the fibres of the neuroglia tissue and become disseminated through the gray columns, for the distance of a number of cord segments, but pushes the neuroglia tissue from it on all sides, stretching and breaking down its fibres and thus forming a pool of blood or perhaps several pools, which pools form clots, that occupy more or less completely the gray columns for a space of perhaps three or four cord segments in close proximity to the bleeding point. The pressure of these clots upon the delicate tissues of the neurons, causes softening and destruction of them. When the mass of blood clot, and dead neurons is absorbed, cavities are left in the cord substance, and the functions of that part of the system presided over by these nerve cells and axones are permanently lost. These cavities in the cord later fill up with connective tissue. The larger the clot the greater will be the destruction of nerve tissue and the greater the loss of function. A small local hemorrhage may produce little or no permanent damage, while a profuse local hemorrhage, will cause permanent loss of function over a large area of the system. In the disseminated hemorrhage the blood makes its way between the neuroglia fibres and about the cells in the gray columns, permeating the substances of these columns for a distance of eight or ten cord segments, but pools of blood do not form, hence there are no clots to produce pressure and destroy nerve tissue. Hence permanent loss of function does not follow this form of hemorrhage, while in the profuse form of disseminated hemorrhage, clots form throughout the disseminated area, and produce pressure on the nerve cells and axones, causing softening and destruction of them, and when these small masses are absorbed numerous small cavities are left in the cord substance, and permanent loss of function occurs over numerous areas of the system more or less remote from each other.

Some of the important things to be remembered in connection with hematomyelia are first, profound general conditions that may follow apparently slight traumatisms applied to the spinal column in such a way as to cause extreme extension or flexion of the cervical segments of the cord. Second, profound conditions involving the cervical cord, due to traumatic insults perpetrated at a distance remote from the cervical region. Third, the cord lesion may appear many days or several weeks after the traumatic insult is received. Fourth, if the hemorrhage is profuse and local, permanent damage to function will result to large contiguous areas in both upper and lower extremities, while if the hemorrhage is profuse and disseminated, permanent damage to function will result in many areas, in both upper and lower extremities more or less remote from each other. If the hemorrhage is small and local, or slight and disseminated, no permanent damage to function will result. Fifth, all forms of hematomyelia improve for about eighteen months, after that little improvement may be expected. Sixth, a guarded prognosis should always be made.

As to the diagnosis of this condition we have no means at our command for making anything more than a probable diagnosis of hematomyelia. Lumbar puncture in suspected cases has been advocated, but as a matter of fact is worse than useless. If blood should be found by this measure in the cerebro spinal fluid, it would not be of diagnostic value, as it would only prove that hemorrhage had taken place into the subarachnoid space, the only portion of the spinal canal, normally occupied by cerebro spinal fluid.

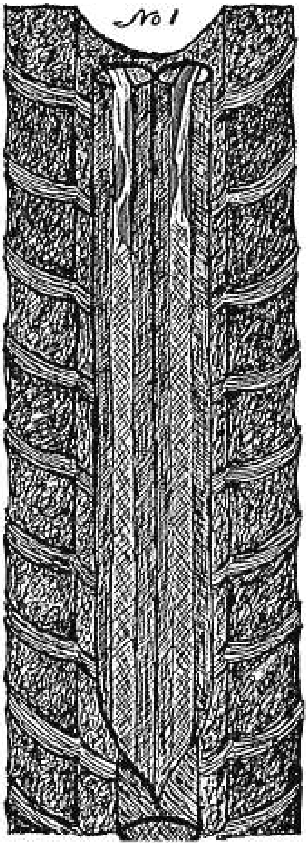
In a hypothetical case, in which paralysis of a portion of the upper and lower extremities, followed immediately a traumatism to the spinal column, remote from the cervical region, the paralysis increasing in extent and severity for several hours after the violence, without cranial nerve involvement. In a youthful patient free from disease, we would be justified in making a diagnosis of hematomyelia, but even in such a case we would think of hysteria as a possible cause of the condition, while in another case less clear, we would have to consider the possibility of scattered foci or morbid tissue changes in the cord due to latent degenerative changes, which have become active by reason of the traumatic insult. As a rule, however, the difficulties are greater in making diagnosis in real cases than in the hypothetical ones. Nevertheless with disseminated inhibition of physical function, in the upper and lower extremities, with increased reflex activity, and exaggerated tremors, following traumatism to the spinal column, more or less remote from the cervical region with no cranial nerve involvement, a diagnosis of hematomyelia is the most probable. If the symptoms of hematomyelia do not appear until a time more or less remote from the date of injury, differential diagnosis is more difficult, for the reason that more time has been provided in which latent degenerative conditions may become active. Even after the lapse of eighteen or twenty months, we may not be able to decide positively what was the true condition.

In slight hemorrhages either local or disseminated the patient will completely recover. The same result will follow hysteria as a rule, hence complete recovery does not decide for hematomyelia. In profuse hemorrhages the patient will improve for a time, but never fully recover. The same may occur in degenerative conditions. Hence we believe in a majority of cases we will not be able to make anything more than a probable diagnosis of hematomyelia, either at the beginning or end of the case.

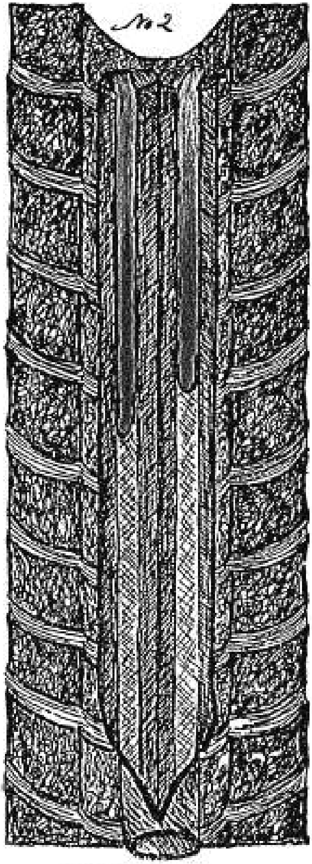
The following illustrates a probable case of hematomyelia, the hemorrhage being postponed, profuse and local.

Mr. J., aged 33 years, teamster by occupation, denies having had any venereal disease or illness of any kind. Patient was thrown from his wagon striking his back, a little to the left of the spinal column, on the curbstone, sustaining a fracture of the seventh and eighth ribs near the column. I saw this patient a few moments after the injury occurred. The fractured ribs were cared for in the usual manner. The patient made a rapid recovery and in six weeks resumed his duties as teamster. Eight weeks after the injury, while caring for his team, preparatory for the day's work, suddenly and without warning, his lower limbs gave way and he settled down in a bunch on the barn floor. Two neighbors who happened to be present tried to assist him to his feet, but found that he had lost the use of both upper and lower extremities. He was perfectly conscious, could talk without difficulty, and knew all that was going on about him. His friends carried him into the house and put him to bed. I saw the patient a half an hour later. Sensation and motion were lost in the entire right lower limb, so profound was the condition in this limb, the patient could not tell where it was unless he could see it. Motion was lost in the left lower limb below the knee and greatly impaired above the knee, but not entirely lost. Sensation in this limb was not completely lost, patient could tell when his left limb was moved and would respond to the prick of a pin. The right arm was paralyzed over the areas supplied by the musculo spiral, median and ulnar nerves, while the left arm was paralyzed over the areas supplied by the musculo spiral and median nerves. There were none of the cranial nerves involved, and the functions of micturition and defecation were at no time involved. Some little improvement in sensation commenced in the area supplied by the ulnar nerve three months after the paralysis occurred. Two months later both arms commenced improving over the entire areas involved. At the end of six months some improvement commenced in the lower limbs. Sensation began to appear in scattered areas over the right leg. At the end of twenty months the patient was able to use his arms in a manner —he could feed himself and partially dress himself, and use a cane in each hand. He had also regained the use of his limbs, so that he could walk in a way. He had better use of the limbs above than below the knees. He walks by raising the limb above the knee and then swinging the lower limb forward. It has now been about thirty-four months since the injury, but there has been no improvement for fourteen months. At the time of the injury the patient weighed 180 pounds. Six months later he weighed 120 pounds and at the present time weighs 140 pounds, but has not gained any in weight for a year. The knee jerk is exaggerated at the present time, while the Argyle-Robertson pupil and Romberg's sign cannot be demonstrated. This patient has never suffered pain, has never had a rise of temperature, and the pulse and respiration have always been normal. Neither has there at any time been tenderness anywhere along the spinal column, or any discoverable abnormality connected with it.

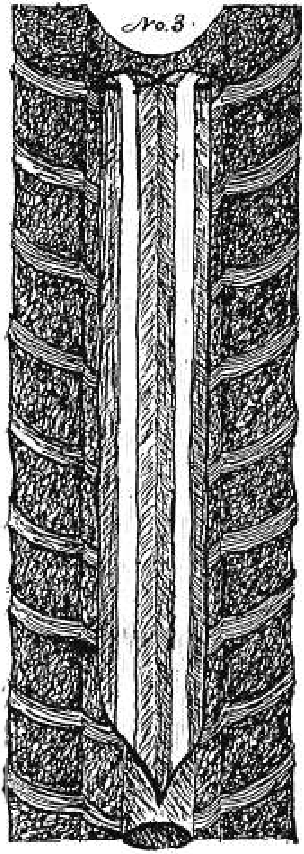
It is impossible for us in this case to positively associate the traumatism with the hemorrhage that occurred at so remote a period. We are well aware that hematomyelia occurs sometimes without traumatism, but in such cases the arteries are diseased. In the case presented there are no evidences of dis-



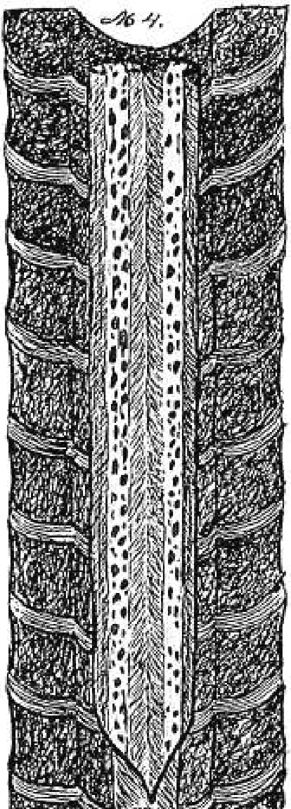
Slight local hemorrhage.



Profuse local hemorrhage.



Slight disseminated hemorrhage.



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Profuse disseminated hemorrhage.

case. We may regard the case from two view points: Was the hemorrhage due to the traumatism, or did it occur independently of it? In other words, would this patient have had his hemorrhage if he had not received the injury? Which is the more reasonable of the two conditions? To me, the former is overwhelmingly impressed as the true one.

It is only comparatively recent that hematomyelia has been recognized as a pathologic entity. For many decades it was one of that group of unknown conditions (except symptomatically) known as railway spine. How many conditions were contained in this group is not known, but it is certain that one of the most important of the group was removed from it when hematomyelia was identified and placed in a category by itself. We now find ourselves facing a new danger; that nearly all cases that were once known as railway spine are now placed, or in danger of being placed, in the category of hematomyelia. As a precaution against this, we can do no better than quote from Leube. "The diagnosis of spinal apoplexy should only be made with the greatest reserve. The sudden onset of the disease, the apoplectiform appearance of paralysis, the most important support for the diagnosis, are also noted in other affections of the cord, especially now and then in acute myelitis. Only when certain etiological conditions simultaneously are in favor of the diagnosis of spinal-cord hemorrhage; a fall, a severe blow upon the back, fracture of the vertebræ, the existence of a marked hemorrhagic diathesis or of a atheroma; a little more certainty in the diagnosis is permissible."

The drawings, Nos. 1, 2, 3 and 4, illustrate a section of the spinal column, with a portion of the arch of each vertebra removed, revealing the cord in the spinal canal. The cord is laid open at the posterior median fissure and each side turned half over showing the hemorrhage in the gray columns in each side of the cord.